USDA FOREST SERVICE RESEARCH PAPER PNW-102
1970

aminated Root Rot
f Douglas-fir
n Western Oregon
nd Washington

By T. W. Childs

PACIFIC NORTHWEST FOREST AND RANGE EXPERIMENT STATION
U.S. DEPARTMENT OF AGRICULTURE FOREST SERVICE

Contents

Introduction

Laminated root rot, caused by the fungus *Poria weirii*, is damaging to many conifers in the Pacific Northwest, and especially to Douglas-fir *(Pseudotsuga menziesii)* west of the Cascade Range in Washington and Oregon. It attacks trees of all ages but is most destructive in stands from about 25 to 125 years old.

It is important primarily as a killer. Young infected trees seldom survive long enough for reduction of growth rate or extension of decay into the butt log to be of much consequence. In old-growth stands, where killing is slower, infection is less common, and decay usually extends only a few feet into butt logs and involves mostly the low-quality core.

This paper summarizes results obtained during the past 20 years from research still in progress.

Description

There are two forms or varieties of the fungus. One is common in the northern Rocky Mountains, principally on western redcedar *(Thuja plicata)*. The other is common in the Pacific Northwest, principally on hosts other than western redcedar. This paper is concerned exclusively with the latter form.

Early stages of *Poria weirii* decay appear as reddish-brown to brown streaks or broad bands on longitudinal sections of roots and trunks, and as circular, crescent-shaped, or irregular areas on cross sections (fig. 1*A*). In later stages of decay the annual rings tend to separate, and the wood contains numerous pockets about a fiftieth of an inch in diameter and a twentieth of an inch long. In final stages, wood breaks down into a loose, stringy mass and eventually may disintegrate completely, leaving butts hollow except for the branch bases, which remain sound (fig. 1*B*).

Figure 1.—*A*, Cross section of Douglas-fir base showing incipient and typical decay caused by *Poria weirii*; *B*, base of tree killed many years ago, showing persistent branch bases.

A

B

Thin, velvety layers or sparse tufts of brown fungus threads are usually present in crevices in decayed wood; and thin, brown crusts without pores sometimes form on surfaces of breaks. Sporophores are brown crusts containing hundreds of minute pores per square inch; they form in root crotches or on lower sides of down trees during late summer and early fall. By the next spring, they are mostly dead and moldy. Although fairly common in some years and localities, they are too inconspicuous to be useful in detecting the disease.

Some infected trees are "rot-thrown"—that is, blown down as a result of destruction of one or two of the main roots—before crown symptoms become conspicuous. Others slowly die standing, from killing of much of the root system or sometimes from girdling at the root collar.

Infection is occasionally found in single trees with no other visibly diseased ones nearby but usually occurs in patches or "centers" of infection (fig. 2), ranging from only a few feet in diameter to more than an acre in extent. Typical centers contain remains of trees, standing or more often down, that have been dead for different lengths of time. Some roots of down trees are broken transversely near the trunk (fig. 3*A*), exposing typical decay. Surviving trees in the interiors of centers are usually leaning or have thin or ragged crowns (fig. 3*B*), poor color, "distress crops" of abnormally small cones, or decreased terminal and lateral growth. Recent rot-throws as well as standing dying trees are often invaded by Douglas-fir bark beetles *(Dendroctonus pseudotsugae)*.

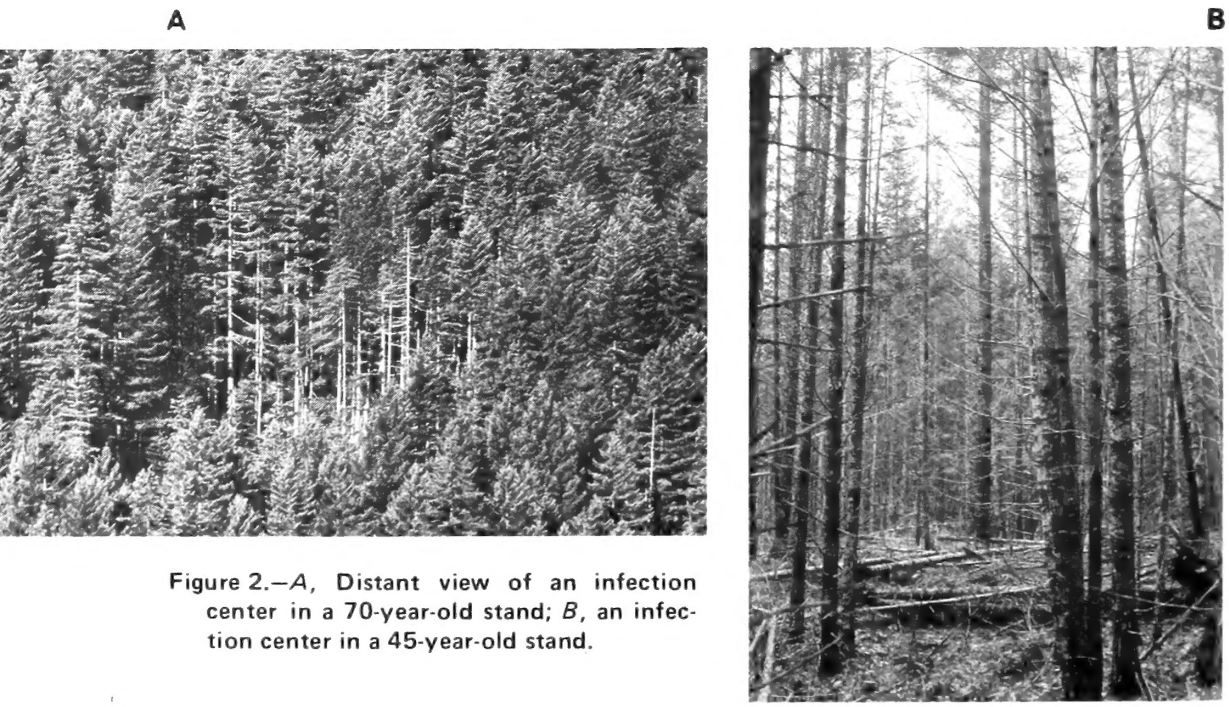


Figure 2.—*A*, Distant view of an infection center in a 70-year-old stand; *B*, an infection center in a 45-year-old stand.

In the interiors of infection centers, much or all of the forest cover may be destroyed over areas of several hundred square feet in pole stands and several thousand square feet in young-mature stands. In outer portions of centers, although forest cover is more or less intact, most of the trees are infected even though no symptoms may be visible; few of them will survive long enough to take advantage of the increase in growing space afforded by the death of their

A Figure 3.—*A*, Douglas-firs rot-thrown by *Poria weirii*, showing stubs of decayed roots; *B*, dead and dying trees in an infection center. B

neighbors. Opening of stands by this disease is a major cause, if not the principal one, of the retrogression from normality reported by Briegleb (1942).[1]

Occasional small centers are found where the disease seems to be no longer active, but irregular spread usually continues more or less indefinitely into the surrounding stand. Infection centers merge so gradually into the surrounding stand, or into nearby centers, that their numbers, dimensions, and distances between them can be determined only approximately.

Small centers are often overlooked, and centers of all sizes are often attributed to other causes. They are, however, readily distinguishable from stand openings caused by other pests and disturbances; and it is important to recognize them since they are usually indicative of continuing damage. In blowdowns and groups of beetle-kills where root rot is not a factor, most of the killing will be found to have occurred simultaneously or nearly so, and roots are mostly sound. Where root rot is caused by *Armillaria mellea*, white to cream-colored mycelial fans[2] are present between bark and wood at bases of dead and dying trees. Less common root-rotting fungi, such as *Polyporus tomentosus* and *Fomes annosus*, cause decay differing in appearance from that caused by *Poria weirii*.

Range and Hosts

Laminated root rot is known to occur from central British Columbia to southern Oregon and eastward into northern Idaho. It is common from sea level to well above the upper limits of commercial forests, on good to poor sites, and on many kinds of soils from deep loams to gravels.

---

[1] Names and dates in parentheses refer to "Literature Cited," p. 26.

[2] These fans are sometimes present in Poria-killed trees saprophytically invaded by Armillaria, but in such instances they are not accompanied by the copious resinosis characteristic of parasitic attack.

Douglas-fir, Pacific silver fir (*Abies amabilis*), lowland white fir (*A. grandis*), and mountain hemlock (*Tsuga mertensiana*) are the most consistently susceptible hosts. Infection centers similar to those in Douglas-fir stands often occur in pure stands of silver fir and in subalpine stands predominantly composed of mountain hemlock.

Western larch (*Larix occidentalis*), alpine fir (*Abies lasiocarpa*), white and lodgepole pines (*Pinus monticola* and *P. contorta*), and Sitka and Engelmann spruces (*Picea sitchensis* and *P. engelmannii*) are attacked when associated with more susceptible species. Ponderosa pine (*P. ponderosa*) and western redcedar are rarely attacked. One probable infection, not verified by culture, has been observed in incense-cedar (*Libocedrus decurrens*).

Western hemlock (*Tsuga heterophylla*) appears to be about as susceptible as any other host where mixed with Douglas-fir of the same age, but is rarely infected in pure stands, and often remains healthy where present as a younger understory in infected Douglas-fir stands. Table 1 summarizes data from a small plot where a hemlock understory had been released during the past century by gradual destruction of the old-growth Douglas-fir overstory by *Poria weirii*. The one hemlock found infected here had grown in immediate contact with a recently rot-thrown Douglas-fir. Similar results were obtained from two plots under similar conditions in the Cascade Range of Washington.[3] Table 2 summarizes data from 20 years of observations on two small plots established in a stand about 50 years old.

Table 1.—Incidence of *Poria weirii* on Douglas-fir and western hemlock on a 0.9-acre plot in the Oregon Coast Ranges

Tree species and condition	D.b.h. class (inches)					
	3-10	10-20	20-30	30-40	40-50	50-60
	Numbers of trees					
Killed by *P. weirii*:						
Douglas-fir	10	24	6	3	1	3
Western hemlock	0	0	0	0	0	0
Living – total:						
Douglas-fir	9	24	13	1	0	0
Western hemlock	28	53	23	5	0	0
Living – infected:[1]						
Douglas-fir	1	3	1	1	0	0
Western hemlock	0	1	0	0	0	0

[1] As indicated by discoloration or decay in increment cores. A few infected trees were undoubtedly diagnosed as uninfected.

[3] Most of the data from all three of these plots were lost in a Station tragedy.

Table 2.—Incidence of *Poria weirii* on Douglas-fir and western hemlock on 3.4 acres of plots in the Oregon Cascade Range

Tree species and condition	D.b.h. class (inches)		
	2-6	6-10	>10
	Numbers of trees		
Killed by *P. weirii* before plot establishment:			
Douglas-fir	23	40	104
Western hemlock	0	0	0
Living when plots were established:			
Douglas-fir	219	75	289
Western hemlock	119	20	0
Killed by *P. weirii* after plot establishment:			
Douglas-fir	23	14	89
Western hemlock	0	0	0

Previous Research

Murrill (1914) described and named the fungus from a collection of the Rocky Mountain form on western redcedar. Overholts (1931) and Baxter (1934, 1953) also published mycological descriptions and compared the fungus with other brown Porias. Buckland, Molnar, and Wallis (1954) discussed differences between the Rocky Mountain and Pacific Northwest forms.

Mounce, Bier, and Nobles (1940) reported the fungus on Douglas-fir and western hemlock on Vancouver Island. They also described cultural characters, incipient and typical decay, and symptoms of the disease. Bier and Buckland (1947) reported extensive damage to young Douglas-fir stands and described spread around infection centers. Buchanan (1948) found infection in 15- to 20-year-old reproduction associated with infected old-growth stumps. In a heavy windthrow following thinning of a stand where infection was common, Wallis (1954) found that most of the down trees were infected.

Results of several important studies were reported by Buckland, Molnar, and Wallis (1954), Wallis and Buckland (1955), Buckland and Wallis (1956), and Wallis and Reynolds (1962, 1965). They found that the fungus survives less than four months in wood blocks buried in unsterilized forest soil, but that it can survive for more than 50 years in dead roots of infected trees, from which it may spread to living trees; that spread from one tree to another can occur through intact bark of uninjured roots; that mycelium grows profusely on western hemlock roots and invades them readily; and that bark beetle populations increase in infection centers. They conclude that no Douglas-firs are immune, although

some appear less susceptible than others; that species mixtures, thinning, trenching, and burning offer little promise of control; and that the disease will probably increase in severity from one rotation to the next.

From clone studies and other evidence, Childs (1963) concludes that root grafts are not necessary for passage of infection from tree to tree, that the fungus can survive in dead roots for nearly 100 years, and that the great majority of infection centers in present-day stands are attributable to vegetative persistence of the fungus in centers that originated in previous stands. Nelson (1964) found that the fungus survived far longer in buried wood blocks in which zone lines formed than in buried blocks without zone lines, and that soil fungi antagonistic to *Poria weirii* were much more common in the latter than in the former.

Surveys and Plot Studies

Surveys consisted of 20- to 100-percent cruises of more than 6,000 acres, of which more that 2,500 acres were examined. This acreage was mostly in sapling to young-mature Douglas-fir types but included some mixed-conifer types both with and without Douglas-fir. The following data were recorded by 2- by 2-chain sampling units: forest type and stocking, size and location of infection centers, and estimated percentage of stand destroyed. Cruise data were occasionally checked by counts of trees living and killed on systematically located small plots, and were supplemented by intensive examination of temporary plots, including some old-growth type, on areas of special interest.

These surveys were not designed to provide a reliable estimate of the regionwide prevalence of the disease, but they yielded considerable information on patterns of infection occurrence.

Surveys referred to most frequently in this paper were as follows:

Snow Creek—244 acres examined in a pure, 27-year-old stand of planted Douglas-fir in eastern Clallam County, Washington, elevation 1,300 feet, site quality III.

Jimmycomelately—179 acres examined in a 70-year-old stand, partly pure Douglas-fir and partly mixed conifers, a few miles north of Snow Creek, elevation 600 to 1,600 feet, site quality IV.

Guler Road—A 115-year-old stand in Skamania County, Washington. Eastern part: 704 acres examined, mostly in mixed conifers, elevation 2,500 to 3,400 feet, site quality IV. Northwestern part: 128 acres examined, partly in pure Douglas-fir and partly in mixed conifers, elevation 2,600 to 2,800 feet, site quality III. Southwestern part: 672 acres examined, pure Douglas-fir, elevation 2,500 to 2,800 feet, site quality III.

To determine rates of damage, we established twenty 10-acre plots in large-sapling to young-mature Douglas-fir stands ranging from very lightly to very heavily infected. Plot locations were determined largely by convenience of access, freedom from roads or other complicating features, and reasonable prospects of freedom from disturbance for a few decades. Plots were fairly representative of the much more extensive stands in which they were situated, except that infection on some of them was more common than in surrounding stands.

Plots were subdivided into 2- by 2-chain blocks. On each block, living trees were tallied by species and d.b.h., and trees killed by Poria were sketch-mapped and tallied by species, d.b.h., and estimated number of years since death. In subsequent examinations, mortality (except suppression in sapling and small-pole stands) was tallied by species, d.b.h., and cause of death, and trees killed by Poria were sketch-mapped. Brief descriptions of 11 of these plots (excluding the most lightly and the most heavily infected and those disturbed by cutting) are given under "Rates of damage."

On a few smaller plots, all living trees and trees that had been killed by Poria were given numbers, stem-mapped by plane table, and recorded by species, d.b.h., and symptoms of disease (or estimated years since death). Deaths from Poria and symptom changes were recorded in subsequent examinations.

Killing by the disease occurs so irregularly that many years of records from numerous plots will be required for accurate determinations of damage rates under different intensities of infection. Records now available provide a basis for only rough estimates.

Since *Poria weirii* appears unable to compete with other fungi in trees where it did not become established prior to death, all dead trees in which it was found were tallied as killed by Poria. Although probably not the immediate cause of death in every instance, it would almost always have killed within a few years. In any event, the slight overestimation of disease impact that may have resulted from errors in assigning causes of death was more than compensated by the relatively small basal areas of killed trees as compared to what their basal areas would have been had they survived until the stand was measured.

Patterns of Infection Occurrence

Poria weirii is a native of the Douglas-fir type. It is just as "normal" an inhabitant as vine maple, but is much more irregularly distributed. On the 51 quarter sections partly or entirely examined during the surveys, impact of the disease varied as follows:

Percent of stand destroyed	Number of quarter sections
Less than 0.1	10
0.1 - 2.0	19
2.1 - 4.0	6
4.1 - 6.0	7
6.1 - 8.0	3
8.1 - 10.0	2
More than 10.0	4

The disease varies greatly in abundance even over short distances in otherwise uniform stands. Like infected trees, infection centers tend to be associated rather than randomly distributed. Infection distribution shown in figures 4 and 5 is characteristic except that infection was more than ordinarily common on these two areas.

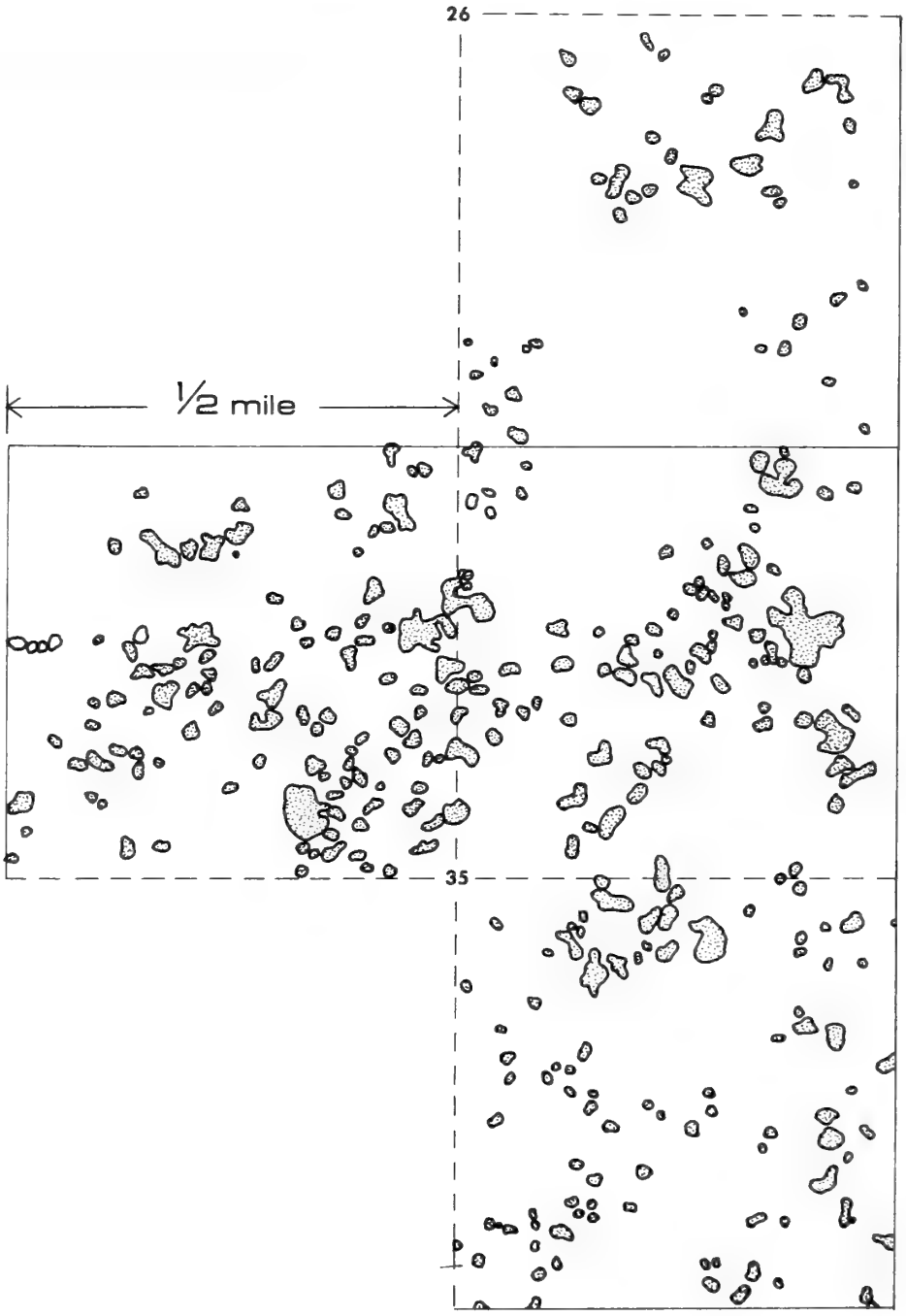


Figure 4.—Infection centers in a pure, well-stocked, and even-aged stand of young-mature Douglas-fir (southwestern part of Guler Road survey). Shaded areas are infection centers or aggregates of closely associated centers.

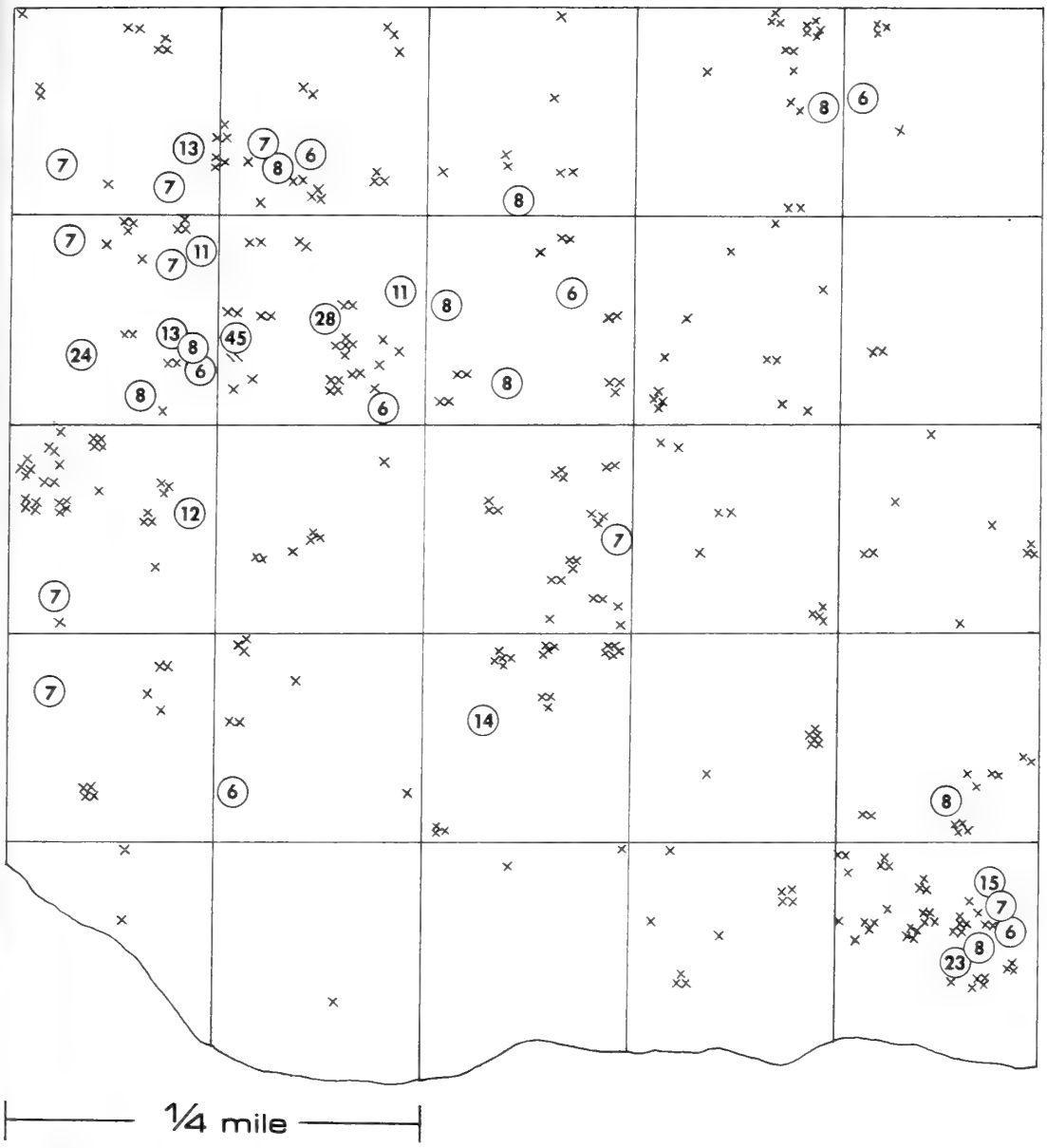


Figure 5.—Infection in a densely stocked plantation of 27-year-old Douglas-fir (Snow Creek survey). Crosses are individual killed trees; circled numerals are numbers of killed trees in groups of more than five.

On the quarter sections shown in figure 4, and on an adjacent quarter section where the stand was also pure Douglas-fir, incidence of the disease was as follows:

Locality	Percent of 2- by 2-chain blocks with infection	Percent of stand destroyed
SW ¼ sect. 26	9	Trace
SE ¼ sect. 26	22	2
NW ¼ sect. 35	55	6
NE ¼ sect. 35	40	6
SE ¼ sect. 35	36	4

In the 70-year-old stand at Jimmycomelately and on the remainder of the Guler Road survey, where stands were less uniform and included considerable proportions of mixed-conifer types, infection incidence by quarter sections ranged as follows:

Locality	Percentage of 2- by 2-chain blocks with infection	Percentage of stands destroyed
Jimmycomelately	56 to 91	6 to 14
Guler Road	0 to 28	0 to 5

Differences are even greater, of course, between smaller subdivisions of land. On 10-acre subdivisions of the area shown in figure 4, percentages of 2- by 2-chain blocks infected ranged from 0 to 76, and percentages of stand destroyed ranged from 0 to 18.

Differences in incidence of infection may be either a result or a cause of forest type differences. In the examples in table 3, types at Guler Road were probably much the same in the early life of the stand as at present. Here, the common occurrence of infection on blocks of "other conifers only" was attributable to high susceptibility of Pacific silver fir, which was especially abundant on section 30, where it formed occasional small pure stands. At Jimmycomelately, western hemlocks and western redcedars were common but usually too small to affect type designations except where death of Douglas-firs had released them. The following regressions (all significant at the 1-percent level) were obtained from 344 sample plots, each one-twentieth of an acre in extent, in this stand:

Dependent variable	Change in dependent variable for each increase of 1 percent in Douglas-fir basal area killed by Poria
	(Percent)
Basal area of living Douglas-fir	-0.7
Basal area of living "other conifers"	+1.3
Basal area of living total conifers	-.4
Number of "other conifers," 4-inch d.b.h. and larger	+.8

At Jimmycomelately, many blocks of "other conifers only" had originally contained Douglas-firs later killed by Poria. At both Guler Road and Jimmycomelately, most infection on blocks of mixed conifers was of Douglas-fir or true firs.

Present differences in infection may be correlated with past differences in infection or in type. Vegetational changes associated with infection centers in preceding stands may have influenced the proportion of highly susceptible species in present stands—for example, by affecting the intensity of terminal fires. As will be shown later, infection centers persist from one forest generation to the next and consequently are most likely to be common where stands of highly susceptible hosts have occurred in uninterrupted succession.

Complex relationships undoubtedly also exist between the disease and stand density, but small dead trees disappear so rapidly that original densities could not be determined. Here, too, conditions in present stands are no doubt

Table 3.—Examples of *Poria weirii* occurrence in different forest types[1]

Locality	Total blocks				Blocks with Poria			
	D[2]	D+ others[3]	Others +D	Others only	D	D+ others	Others +D	Others only
	Number				*Percent*			
Guler Road:								
E 1/2 of sect. 30	331	228	40	127	5	12	30	46
Sects. 19, 20, and 29	207	565	116	55	3	2	1	2
Remainder	86	163	39	45	16	18	10	7
Jimmycomelately	319	100	20	4	74	79	85	25

[1]Data are from only those quarter sections where both pure Douglas-fir and other coniferous types were present.

[2]Douglas-fir.

[3]Other conifers.

partly attributable to those in preceding stands. Interaction of *Poria weirii* and its environment is a fertile field for hypotheses but is unlikely to yield many definite conclusions until a larger factual basis is available.

Number of centers, as well as their distribution and area, is important because further damage will result largely from spread into the surrounding stand and will consequently be greater from numerous small centers than from a few larger ones with more total area but less periphery. On the 640 acres of the young-mature stand at Guler Road shown in figure 4, there were 342 centers of the disease averaging 0.1 acre in size, and about 4.4 percent of the stand had been destroyed. On the 244 acres of the 27-year-old plantation at Snow Creek shown in figure 5, there were 199 centers averaging less than 0.01 acre in size (often consisting of only one or two dead trees), and less than 1 percent of the stand had been destroyed, but presence of infection at so many places will probably result in losses per acre exceeding those at Guler Road before the plantation reaches commercial maturity.

Although control of laminated root rot is not yet possible, losses can often be reduced if incidence of the disease in the various parts of the stand is known. Since the disease is so irregularly distributed, ground surveys accurate enough to be useful are too expensive to be generally practical at this time, even when made in conjunction with surveys for other purposes. Medium-sized to large infection centers and concentrations of small ones were easily visible on 1:12,000 aerial photos of the area shown in figure 4; but even on 1:3,000 photos they could not be distinguished from old beetle-kill areas, brush patches, and other stand openings not indicative of continuing losses. Until better methods become available, information needed for good management must be obtained by careful observation, at every opportunity, of disease conditions in all parts of the stand.

Persistence and Spread

Although *Poria weirii* produces great numbers of viable spores, it appears unable to establish itself in dead material in competition with other fungi. Examinations of thinnings, some more than 30 years old, have disclosed no instance of stump infection by spores. Only one doubtful instance has been observed of infection of Douglas-fir through a trunk wound, and very few infections through wounds have been found in hundreds of trees of other species dissected in decay studies.[4] From studies of clones of the fungus it is evident that the great majority of infection centers now existing are "holdovers"—that is, results of vegetative persistence and spread of infections that started from spores in stands preceding the present ones.

Tests with hundreds of cultures of *Poria weirii* have shown that, if moisture and nutrients are adequate for vigorous growth until contact between mycelia is well established, all isolates of a clone are compatible in culture with all other isolates of that clone and antagonistic[5] to all isolates of other clones (fig. 6). Since *P. weirii* does not produce asexual spores, isolation of the same clone from different trees is proof that spread of the fungus between them has not been by spores. Transfer of vegetative inoculum by insects or other vectors is possible;

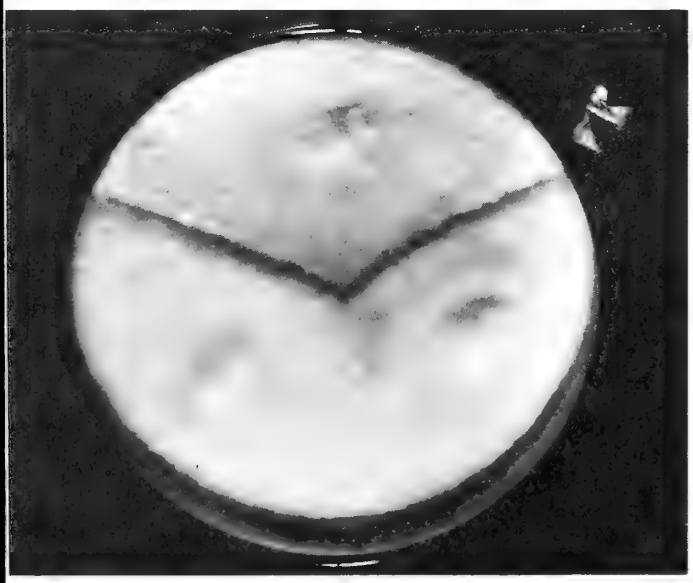


Figure 6.—Three isolates of *Poria weirii* growing in the same petri dish. The two isolates that have grown together with no indication of antagonism are of the same clone, from trees about 65 feet apart; the isolate that is separated from the other two by a narrow dark zone is of another clone, from a tree about 375 feet from the first two.

[4] Buckland, Foster, and Nordin (1949) found no *Poria weirii* in 719 dissected Pacific silver firs and only two infections through wounds (six others from undetermined entry points) in 963 western hemlocks. Wright and Isaac (1956) found four infections associated with wounds in 1,030 western hemlocks and none in 126 Sitka spruces and 62 true firs.

[5] Buckland, Molnar, and Wallis (1954) found no antagonism between isolates from Douglas-fir but do not state whether their isolates were from closely associated or widely separated trees.

but studies of mycelial growth on roots (Wallis and Reynolds 1962, 1965), of the close association of diseased living trees with killed trees (Childs 1963), and of orientation of decay on tops of stumps (to be discussed later) leave little doubt that clonal spread is almost always, if not invariably, by vegetative growth of the fungus.

Tests of isolates from 152 infection centers, on 10 of the 10-acre damage plots in six stands, showed that 130 of the centers were caused by only 36 clones, each of which was isolated from two or more centers. Occurrence of the same clone in distinctly different centers cannot have resulted from vegetative spread during the life of the present stand, because centers of the same clone are separated by zones of healthy forest often more than 100 feet wide. Examples of clones occurring in more than one center are shown in figure 7.

Of the 22 centers occupied by clones not found elsewhere, probably few if any originated from spore infections in present stands. Ten of the 22 were in corner blocks of plots, and only four were more than 2 chains from plot edges, so several of them undoubtedly had clones in common with centers off the plots (or with plot centers from which no cultures were obtained). The shape of some suggests formation by fusion of smaller centers. And since 10 of the 36 "multiple-center" clones were found in only two centers each, and 11 in only three centers each, it can reasonably be assumed that several "holdover" clones have appeared in only one center each.

Infection from residues of the previous stand at more than one point within a center is probably fairly common, but most spread is between trees of the present generation. This is evident not only from the close association of recently killed trees with older kills (fig. 8), and the rapid decrease in percentage of living trees infected as distance from killed trees increases (Childs 1963), but also from the tendency of decay in living infected trees to be oriented toward the nearest killed tree. On a 16-acre clearcut area at Guler Road, 149 stumps with unsymmetrical decay caused by *Poria weirii* were found within 50 feet of killed trees; 101 of these 149 had the most extensive decay within 45° of the direction of the nearest killed tree, and only 20 within 45° of the opposite direction. Since some trees undoubtedly become infected by indirect routes, some from nearby but not the nearest killed trees, and some from infected trees not yet killed, it is evident that most infections result from tree-to-tree spread.

Rate of vegetative spread from tree to tree cannot be determined from data now available, since instances of apparently rapid spread may or may not be results of new infections from residues of the previous stand and since groups of progressively infected trees are often rot-thrown simultaneously or may die almost simultaneously when subjected to unusual moisture stress. Spread rate probably varies widely in response to differences in root network patterns and other vegetational and soil factors. Figure 8 shows that spread is far from uniform around centers even where susceptible hosts are about equally numerous in all directions.

The fungus grows fairly rapidly along root surfaces (Wallis and Reynolds 1962) but increase in average radius of infection centers appears to be less than a foot per year. Nevertheless, long-continued vegetative spread (in some instances probably during several forest generations) often distributes infection over considerable distances (table 4).

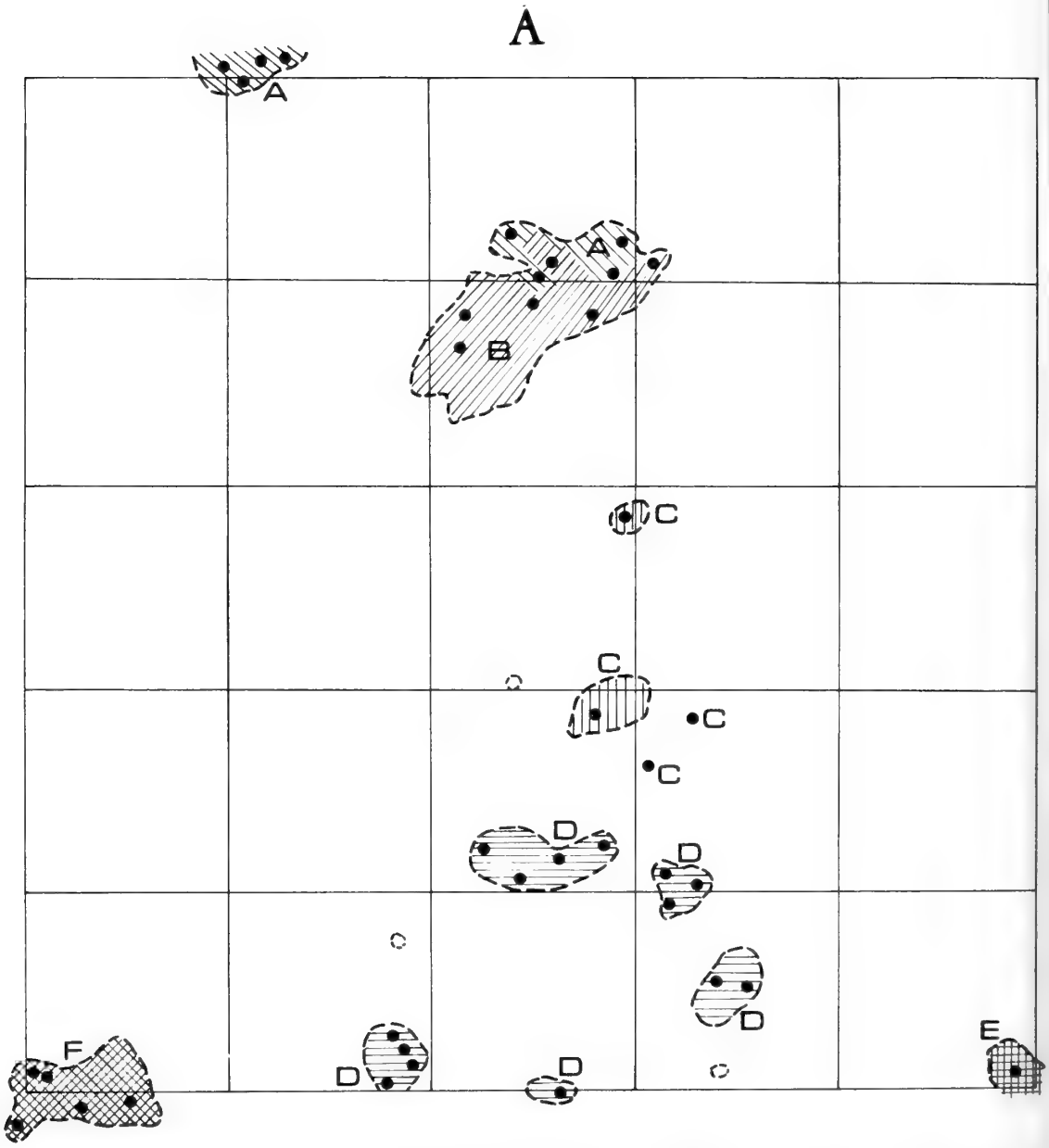
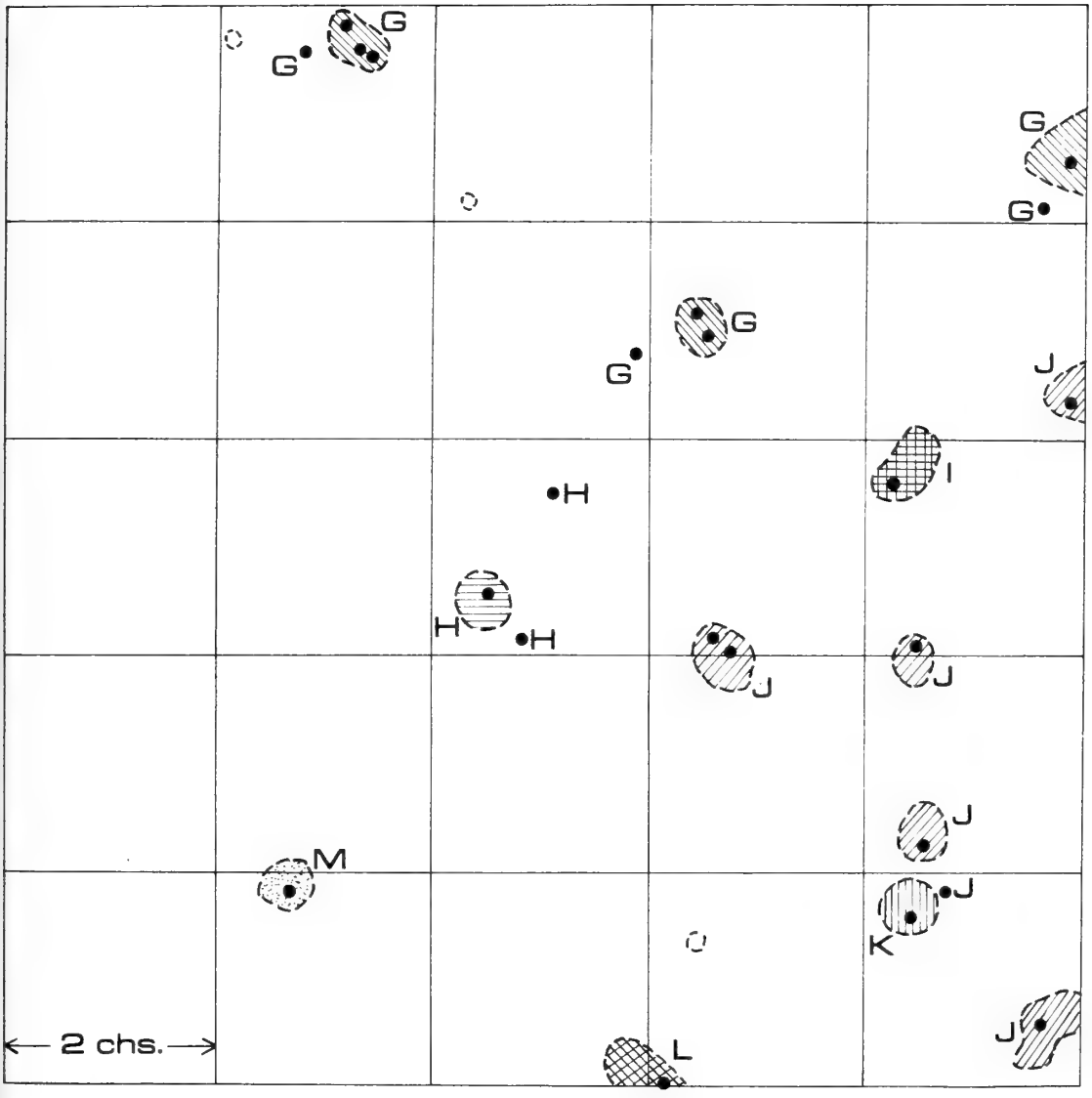


Figure 7.—Clones and infection centers on: *A*, Clemons south plot and *B*, Snow Creek east plot. Broken lines are approximate boundaries of infection centers. Letters are

B



clone designations. Solid circles indicate trees from which cultures were obtained. Unshaded centers are those from which no cultures were obtained.

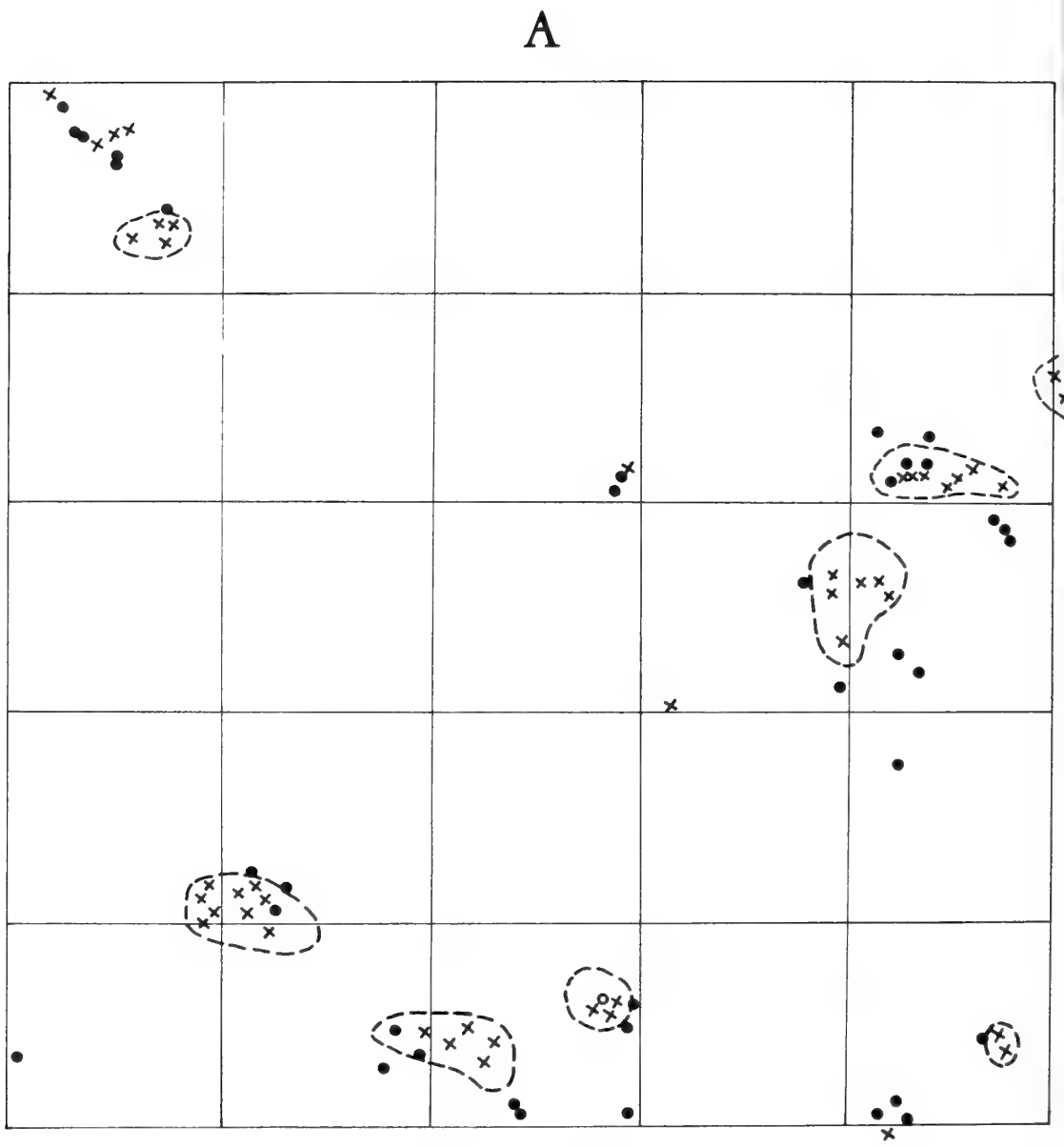
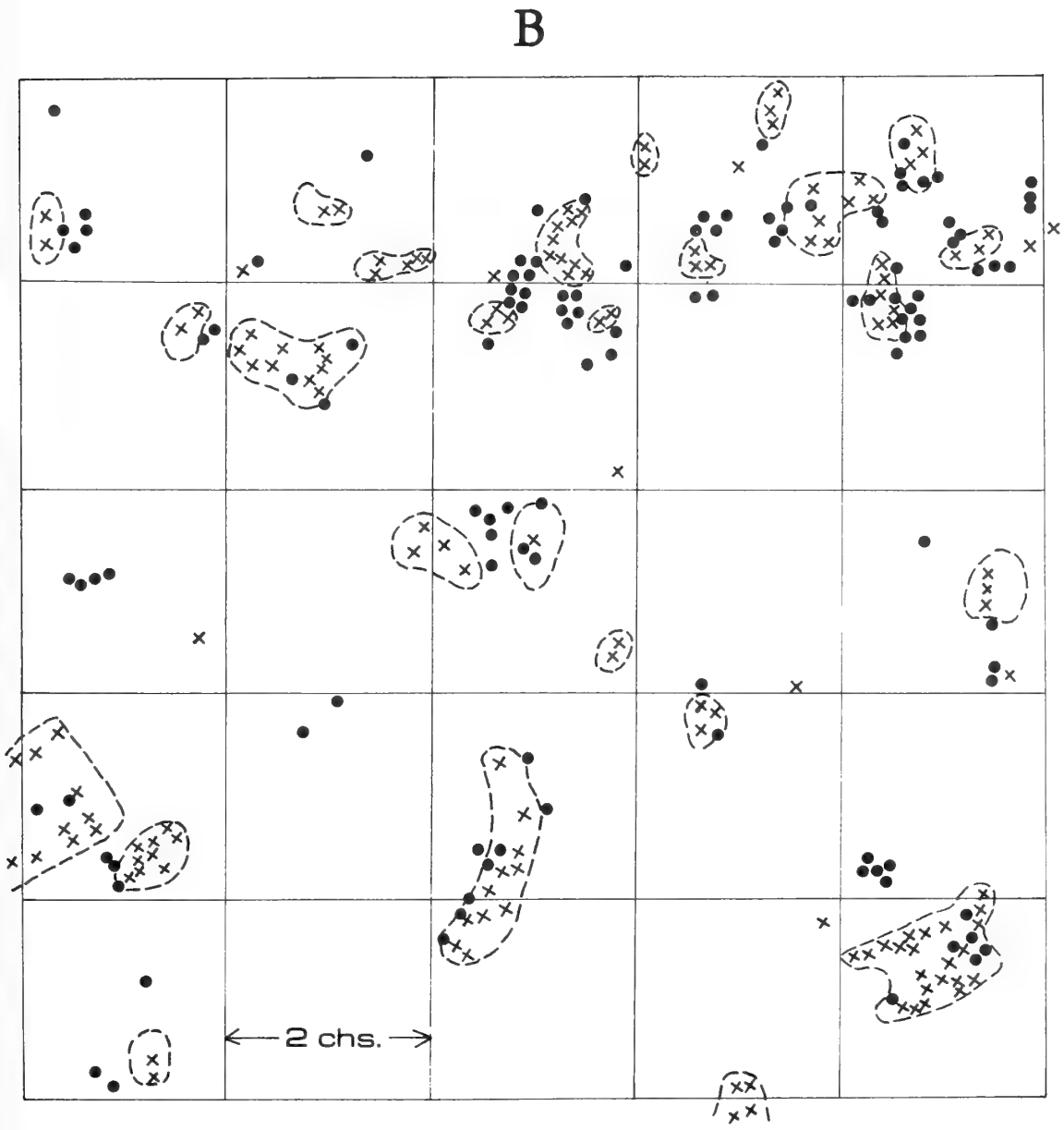


Figure 8.—Association of recently killed trees with previously killed trees at: *A*, River Road plot and *B*, Martha Flat plot. Circles are trees killed since plot establishment (11



years ago at River Road and 16 at Martha Flat). Crosses are trees killed before plot establishment. Broken lines are approximate boundaries of infection centers when plots were established.

Table 4.—Maximum distances between sources of cultures of single clones found in more than one infection center

Plot	Age of stand	Number of centers in which the clone was found						
		2	3	4	5	6	7	8
	Years	*Feet*						
Snow Creek east	28		80	480		390		
Snow Creek west	28		160		330			
Elma	30	290						
Martha Flat	42	70	230	320	140			340
			90					
Martha Creek	43	110	100			350	390	
River Road	50	120	110					
		210	150					
		80	160					
Clemons south	60	280		160	240			
Clemons north	60	100	100	300	460			
			130					
Mouse Creek south	110		400	230		370		
Mouse Creek north	110	520		430				
		220						

Because of its persistence in underground inoculum, *Poria weirii* will probably become more damaging in stands managed intensively—where the new forest generation is established immediately after removal of the old—than in unmanaged stands, where restocking is often delayed.

Damage

In sapling and pole stands, losses of actual volumes are seldom great, and damage consists almost entirely of reduction of productivity through conversion of numerous small areas into brush patches or groups of younger trees of less susceptible species. When changes of the latter kind occur early in the life of the stand and on sites permitting good growth of the less susceptible species, damage may be slight. But on most Douglas-fir sites, especially when the type change is long delayed, productivity is reduced even if forest cover is maintained almost continuously.

The best measure of past damage and indicator of future damage would be numbers and sizes of infection centers if it were practicable to determine these with sufficient accuracy. Percentage of trees killed is only roughly indicative of infection severity, since remains of small killed trees soon disintegrate or are hidden by litter; this percentage is also distorted by irregularities in stand density and normal decrease in numbers of trees as stands become older. Basal area percentage underestimates impact of the disease, since basal areas of trees killed several years ago do not adequately represent, in proportion to present basal areas of surviving trees, their potential contribution to yield. In young stands, the

most important question is how much damage is to be expected before commercial maturity is reached, rather than how much has already occurred; and percentage of either trees or basal areas killed is as good a basis as is now available for approximate predictions.

In young-mature stands most of the damage may have already occurred, but characteristic infection centers are common and substantial losses often continue.

At Guler Road, for example, the disease appeared to have become inactive in some of the smaller centers but was still causing damage in the remainder. On 16 acres of a clearcut area (near the center of fig. 4) there were 1,455 Douglas-firs, living or beetle-killed within the past year, with a basal area of 3,317 square feet. *Poria weirii* had killed 242 trees (14 percent of total living and killed) with a basal area of 479 square feet (13 percent). About half of the killing by Poria had occurred within the past decade. Of the living and recently beetle-killed trees, 279 (19 percent) with basal area of 677 square feet (20 percent) showed *P. weirii* decay on the stump surface and would probably have been killed within the next two or three decades.

Distribution of the disease on this area (fig. 9) suggests that most of the *Poria weirii* clones present had originally appeared in two or more centers each,

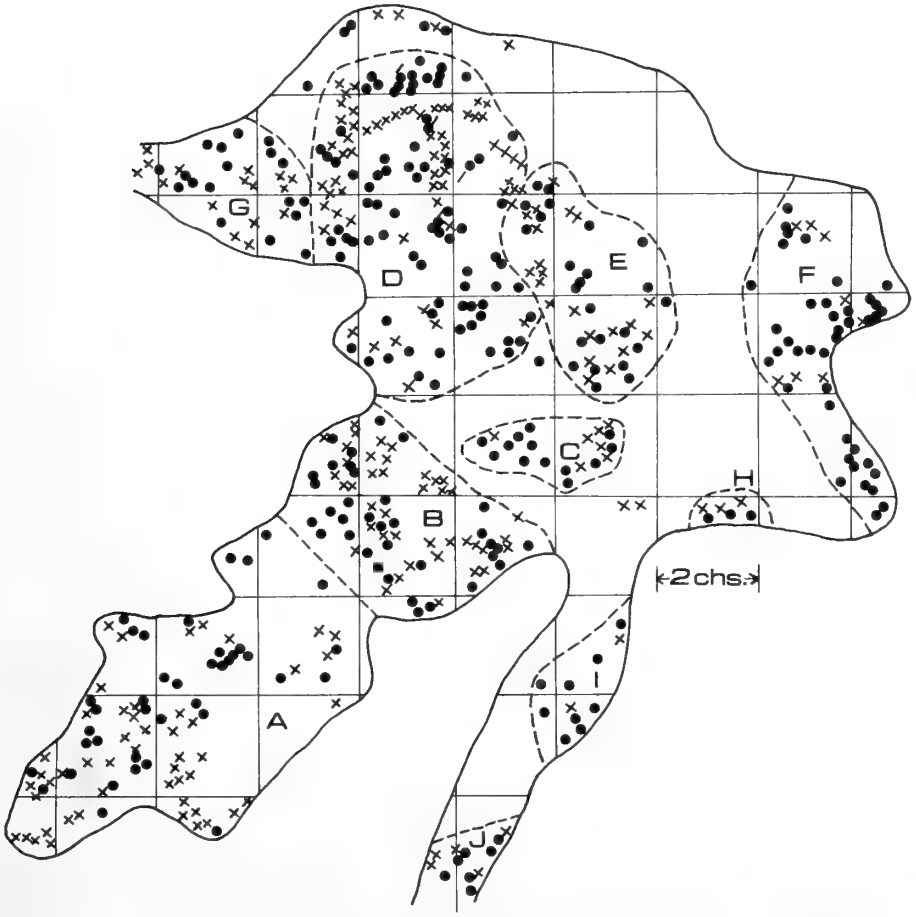


Figure 9.—Distribution of clones of *Poria weirii* and infected trees on 16 acres of a clearcut area. Crosses are trees killed by *P. weirii;* circles are infected living (including recently beetle-killed) trees; letters designate the 10 clones responsible for the damage; dotted lines are approximate boundaries of clone areas.

separated by zones of healthy forest, and that the centers expanded toward each other until they became more or less continuous.

Severe damage by bark beetles in this locality may also have resulted indirectly from the widespread infection by *Poria weirii*. Ordinarily, a large proportion of Douglas-fir bark beetle attacks occur in trees weakened by root diseases. In 1952-53, a period exceptionally favorable for beetles, only 8 percent of trees with Poria infection (as indicated by decay on the stump surface) were killed by beetles on the area shown in figure 9, while 15 percent of the apparently uninfected trees were killed, but over an area of several square miles the killing was obviously concentrated on and near the areas of heaviest infection. This substantiates previous observations that root rot centers help to maintain beetle populations at moderate levels during ordinary years and serve as sources of infestation of surrounding healthy stands when conditions permit beetles to invade uninfected trees.

Larger killed trees in young-mature stands retain some salvable volume for several years, but much of their volume and most of their quality is in the sapwood, which deteriorates rapidly after death.

Even where infection is abundant, cull from *Poria weirii* decay in living trees is of relatively little importance. In a 110-year-old stand, where infection was heavy on 3 acres of the logged area and light to moderate on the remainder, such cull did not exceed 0.5 percent:

	Area A	Area B	Area C
Acres logged	3	25	22
Percent of live stand cut	100	25	15
Percent of total stand killed by Poria	14	4	2
Living trees felled:			
Total number	147	531	359
With Poria decay on stumps:			
Percent by number	24	13	7
Percent by volume	26	12	5
Percent of total felled volume cull from Poria decay	.4	.5	.1

Heaviest losses here were from windthrow subsequent to logging. Only 23 trees went down during the first winter after the cutting, but during the second winter nearly half of the remaining trees on area *B* and about three-fourths of those on area *C* were windthrown.

In old-growth stands, characteristic infection centers are seldom common even where infection is present. For example, no rot-thrown trees could be found on the clearcut area summarized in table 5, and no indications of the disease were visible in the contiguous uncut stand. However, the presence of more hemlock than is usual in this locality may have resulted from killing of some of the Douglas-fir when the stand was young.

When active infection centers do occur in old stands, they are of special concern because of the value of the timber involved. On one center covering a little more than an acre in a 225-year-old stand, approximately 35,000 board feet of Douglas-fir had been killed, 60,000 showed Poria decay on stump surfaces and would presumably have been lost if harvest had been long

Table 5.—Occurrence of *Poria weirii* infection in an old-growth stand with no external indications of infection

Tree species and condition	D.b.h. class (inches)				
	21-30	31-40	41-50	51-60	Total
	Numbers of trees				
Douglas-fir—living trees:					
Total	7	13	22	18	60
Infected[1]	0	2	9	5	16
Douglas-fir—snags:					
Total	3	7	6	2	18
Infected[1]	1	3	1	1	6
Western hemlock—living trees:					
Total	53	38	20	4	115
Infected[1]	4	4	0	1	9
Western hemlock—snags:					
Total	2	1	0	0	3
Infected[1]	1	0	—	—	1

[1] As indicated by decay on stump surface.

postponed, and only 52,000 appeared to be free from infection.

In the most severely damaged old stand examined, data were taken from thirteen 2.4-acre sample plots scattered over several thousand acres. On these plots there were 1,193 living Douglas-firs (basal area 9,182 square feet) and 342 living western hemlocks (basal area 908 square feet). *Poria weirii* had killed 231 Douglas-firs and three hemlocks, or 16 percent of the total living and killed Douglas-firs (12 percent of the basal area[6]) and 1 percent of the hemlocks. The plots were located where infection was obviously abundant, but local foresters estimated average current losses from the disease at nearly 200 board feet per acre per year over an area of about 100,000 acres.

Douglas-fir heartwood decays rather slowly, and large dead trees may remain merchantable for many years, especially if lying on moist ground in shaded situations. They steadily lose value, however, partly from continued decay by *Poria weirii* in butts but principally from decay of the high-quality sapwood and outer heartwood by other fungi. In the stand mentioned in the preceding paragraph, values were being lost so rapidly that logging plans were drastically revised to give priority to the more severely infected areas.

[6] In this instance, the smaller average basal area of killed trees is probably due less to continued growth by surviving trees than to underestimation of original diameters of down trees.

Much of the killed volume can often be salvaged where damage is concentrated. Volumes in occasional trees killed in old-growth stands may be greater in the aggregate and are more difficult to salvage. But usually, by the time the stand has reached the old-growth stage, most of the damage has already occurred, through destruction of immature growing stock and conversion of parts of the stand to brush patches or groups of younger trees of less susceptible species.

Rates of damage cannot be determined accurately from the meager data now available, but rough estimates are possible. Theoretical reconstructions of stands as they existed in past years indicate that damage in young stands doubles about every 15 years (Childs 1960), and records from several years of observations on permanent plots substantiate this conclusion. But ratios of current damage to total damage usually decrease after a few decades.

Table 6 summarizes 1,190 acre-years of records from 11 plots (including five pairs, each separated by several hundred feet) representative of six practically pure Douglas-fir stands. Although these stands differ considerably in site quality and other characteristics, total damage to date of plot establishment (columns 8 and 9) is shown to increase fairly consistently with increasing age. Except on the oldest plots, current annual damage (column 12) also increases with age, largely if not entirely because of increased numbers and peripheries of infection centers. Ratio of current to past damage (column 13) decreases with increasing stand age, as new centers appear less frequently and as enlargement of older centers reduces their periphery-area ratio.

Current damage rates in table 6 may, at first glance, appear too small to be of much consequence, but it must be remembered that these are *annual* losses and that, since most of the stand in infection centers is destroyed, percentage of productive acreage lost is approximately equal to percentage of basal area lost. For example, annual loss of 0.12 percent of basal area (average for the three youngest stands in table 6) over a 60-year period will reduce final yield by about 7 percent. It is estimated that loss from *Poria weirii* in western Oregon and Washington amounts to 32 million cubic feet annually (Childs and Shea 1967); most of this is loss of potential yield from young Douglas-fir stands.

Laminated root rot is not a catastrophic disease and usually not very spectacular. It occasionally causes heavy losses of merchantable timber, but its principal effect is its gradual attrition of growing stock by innumerable small centers of infection.

Further Research Needed

Although dissemination of *Poria weirii* by spores should not be entirely ignored, the obvious major problem is how to retard spread of established infection centers and prevent their persistence from one rotation to the next. Existence of natural controls is shown by the common occurrence of belts of healthy forest between centers inhabited by the same clone of the fungus, and by occasional failure of the disease to continue its spread from small centers. Evidence now available suggests that the principal natural controls are hyperparasitic or otherwise antagonistic soil micro-organisms. Accordingly, studies most likely to yield results useful in forest management appear to be

Table 6.—Total and current damage by *Poria weirii* on 11 lightly to moderately infected plots

Plot[1]	Length of record	Site quality	Stand age[2]	Average d.b.h.[2]	Average stand per acre[2]		Killed by Poria				Current annual loss of basal area from Poria	
							Before plot establishment		After plot establishment			
					Trees	Basal area	Trees	Basal area	Trees	Basal area	Total average[3]	For each 1 percent killed before plot establishment[4]
1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.
	Years		*Years*	*Inches*	*No.*	*Sq. ft.*	----------	----------	*Percent*	----------	----------	----------
Snow Creek west	11	III	28	7.6	547	182	0.47	0.34	0.51	0.81	0.074	0.214
Snow Creek east	11	III	28	7.6	529	182	.83	.50	.72	1.03	.094	.186
Bare Mountain south	6	III	30	8.0	284	116	.66	.46	.25	.16	.026	.057
Bare Mountain north	5	III	30	9.5	214	127	1.02	.97	.70	.65	.130	.135
Martha Flat	16	IV	42	6.5	501	142	2.40	1.79	2.42	2.54	.159	.089
Martha Creek	14	IV	43	6.7	539	155	4.31	3.64	2.67	3.56	.255	.070
River Road	11	II	50	17.7	118	217	3.29	2.77	3.32	3.48	.316	.114
Clemons south	9	II	60	15.3	138	197	6.14	3.57	2.18	2.06	.229	.064
Clemons north	9	II	60	16.7	96	166	6.21	3.96	1.45	1.11	.123	.031
Mouse Creek south	14	IV	110	19.0	112	236	4.91	3.94	1.78	1.78	.127	.032
Mouse Creek north	13	IV	110	16.6	156	247	5.74	4.71	1.67	1.71	.132	.028

[1] River Road plot is in Clackamas County, Oregon. Others are in Clallam, Grays Harbor, and Skamania Counties, Washington.

[2] At time of plot establishment. Douglas-firs only.

[3] Column 11 divided by column 2.

[4] Column 12 divided by column 9.

(1) identification of micro-organisms most effective in control, (2) determination of their environmental requirements, and (3) development of practicable methods for modifying environments to increase the micro-organisms' effectiveness.

Other investigations may contribute to our understanding of the disease or disclose relationships that make it more vulnerable to control. For example, why does western hemlock sometimes remain free from infection in active centers? Is it because of differences in root network patterns? genetic differences between local populations of hemlock? genetic differences between clones of the fungus? Why is the disease usually less damaging to old growth than to young growth? and why is it severely damaging in a few old-growth stands? Why are some trees killed physiologically (by parasitic attack on most of the living tissues of the root system) and others killed mechanically (by destruction of root and lower-trunk heartwood, with living tissues only moderately impaired)? To what extent do soil texture, moisture, and chemical composition directly affect survival of the fungus in dead material?

Such studies should be accompanied by others whose empirical results may be more immediately useful. For example, how is the disease affected by stand density? by various species mixtures, especially with hardwoods? Does early thinning disrupt the root network enough to retard spread of infection? Does inoculum survive as long in young-mature Douglas-fir roots, or in true fir roots, as in the massive roots of old-growth Douglas-fir?

Last but far from least, we need (1) accurate determinations of spread and damage rates, to provide a sound basis not only for the yield calculations essential for good management but also for economic analysis of cost-benefit ratios of control, and (2) techniques for differentiating, at acceptable cost, between disease hazard zones within stands, so that management procedures can be modified wherever such modification will be profitable.

Management Recommendations

Serious damage by laminated root rot in young stands on severely burned areas is conclusive evidence that slash-burning is not effective in control. A few infected root systems may be partly burned out, but most of the inoculum is too far underground to be affected by the fire. By decreasing the organic content and temporarily increasing the alkalinity of the soil, fire may even be somewhat adverse to natural controls.

Direct control methods now available are too expensive and offer little prospect of success. Trenching has proved unreliable for preventing spread of *Armillaria mellea* and *Fomes annosus*, and Wallis and Buckland (1955) found that new centers of *Poria weirii* infection continued to appear outside trenched centers. Although probably only the larger roots are long effective as reservoirs of infection, removal of these would usually require excavation to depths of 3 feet or more. Under field conditions, it is impossible to get penetration by chemical sterilizers into large, deeply buried roots whose interiors are often protected by layers of resin-impregnated wood.

Nevertheless, because of the tendency of laminated root rot to occur in

concentrations, judicious practice of "forestry by the acre" can reduce its economic impact:

1. Become familiar with the disease. Some infection is present in most Douglas-fir stands, and until better quantitative data are obtainable, forest managers must rely largely on personal judgment to determine where it is serious enough to require consideration in management.

2. Where infection is light, ignore it. The costs of excluding a few small centers from standwide operations are greater than the damage to be expected from them.

3. Do not waste money on precommercial thinnings where infection is abundant. Final yield will be considerably reduced by spread of the disease before commercial maturity is reached. Sometimes it may be possible to obtain a good crop by favoring species less susceptible than Douglas-fir, but even the less susceptible species are often infected when associated with highly susceptible ones.

4. When cutting in heavily infected stands, either clearcut or take only dead and dying trees. Infected trees are not windfirm, and partial cuts will be followed by heavy windthrow. Since dead trees give only slight protection to their neighbors, and trees with conspicuous crown symptoms are unlikely to live long, both can be removed without seriously impairing the stability of the stand.

5. Give logging priority to areas where the disease is most abundant – partly to salvage as much of the killed material as possible but principally because such areas are contributing little or no net growth. For example, lightly infected "forties" of the area shown in figure 4 were still making a net growth of 600 to 700 board feet per acre per year while heavily infected ones were losing volume.

6. Do not plant highly susceptible species where infection was abundant in the logged stand (as, for example, on the area shown in fig. 9). Although brush is usually well established on such areas and occupies them rapidly after logging, the certainty of damage to the new stand makes planting too risky an investment. Furthermore, when natural reforestation is delayed, as it often is, spread of infection to the new stand is reduced by death of the fungus in some of the old roots. Immediate planting, in contrast, increases the number of opportunities for growing roots to come in contact with infectious material.

Literature Cited

Baxter, Dow V.

1934. Some resupinate polypores from the region of the Great Lakes. V. Papers Mich. Acad. Sci., Arts, & Letters 1933(19): 305-332, illus.

———

1953. Some resupinate polypores from the region of the Great Lakes. XXIV. Papers Mich. Acad. Sci., Arts, & Letters 1952(38): 115-123, illus.

Bier, J. E., and Buckland, D. C.

1947. Relation of research in forest pathology to the management of second growth forests. I. *Poria weirii* root rot, an important disease affecting immature stands of Douglas fir. B.C. Lumberman 31: 49-51, 64, 66, illus.

Briegleb, P. A.

1942. Progress in estimating trend of normality percentage in second-growth Douglas-fir. J. Forest. 40: 785-793.

Buchanan, T. S.

1948. *Poria weirii*: its occurrence and behavior on species other than cedars. Northwest Sci. 22: 7-12.

Buckland, D. C., Foster, R. E., and Nordin, V. J.

1949. Studies in forest pathology. VII. Decay in western hemlock and fir in the Franklin River area, British Columbia. Can. J. Res. C, 27: 312-331, illus.

———, Molnar, A. C., and Wallis, G. W.

1954. Yellow laminated root rot of Douglas fir. Can. J. Bot. 32: 69-81, illus.

——— and Wallis, G. W.

1956. The control of yellow laminated root rot of Douglas fir. Forest. Chron. 32: 14-19.

Childs, T. W.

1960. Laminated root rot of Douglas-fir. U.S. Dep. Agr. Forest Pest Leafl. 48, 6 pp., illus.

———

1963. *Poria weirii* root rot. Phytopathology 53: 1124-1127, illus.

——— and Shea, K. R.

1967. Annual losses from disease in Pacific Northwest forests. Pacific Northwest Forest & Range Exp. Sta. USDA Forest Serv. Resource Bull. PNW-20, 19 pp.

Mounce, Irene, Bier, J. E., and Nobles, Mildred K.
1940. A root rot of Douglas fir caused by *Poria weirii*. Can. J. Res. C, 18: 522-533, illus.

Murrill, William A.
1914. An enemy of the western red cedar. Mycologia 6: 93-94, illus.

Nelson, Earl E.
1964. Some probable relationships of soil fungi and zone lines to survival of *Poria weirii* in buried wood blocks. Phytopathology 54: 120-121, illus.

Overholts, L. O.
1931. Diagnoses of American Porias. III. Some additional brown species, with a key to the common brown species of the United States and Canada. Mycologia 23: 117-129, illus.

Wallis, G. W.
1954. Commercial thinning in Douglas fir in relation to control of *Poria weirii* root rot. Can. Dep. Agr., Div. Forest Biol. Bi-monthly Progr. Rep. 10(3): 4.

_____ and Buckland, D.C.
1955. The effect of trenching on the spread of yellow laminated root rot of Douglas fir. Forest. Chron. 31: 356-359.

_____ and Reynolds, G.
1962. Inoculation of Douglas fir roots with *Poria weirii*. Can. J. Bot. 40: 637-645, illus.

_____ and Reynolds, G.
1965. The initiation and spread of *Poria weirii* root rot of Douglas fir. Can. J. Bot. 43: 1-9, illus.

Wright, Ernest, and Isaac, Leo A.
1956. Decay following logging injury to western hemlock, Sitka spruce, and true firs. U.S. Dep. Agr. Tech. Bull. 1148, 34 pp., illus.

Childs, T. W.

1970. Laminated root rot of Douglas-fir in western Oregon and Washington. USDA Forest Serv. Res. Pap. PNW-102, 27 pp., illus. Pacific Northwest Forest & Range Experiment Station, Portland, Oregon.

This important killer of Douglas-fir and associated species varies greatly in abundance, even over short distances. Most infection in present stands results from vegetative persistence of the fungus (*Poria weirii*) in dead roots of preceding stands. Most spread from tree to tree is by vegetative growth along the roots. Old-growth trees sometimes are infected, but damage consists principally of destruction of immature growing stock. Control is not yet possible, but economic impact can be reduced by the management practices recommended.

Childs, T. W.

1970. Laminated root rot of Douglas-fir in western Oregon and Washington. USDA Forest Serv. Res. Pap. PNW-102, 27 pp., illus. Pacific Northwest Forest & Range Experiment Station, Portland, Oregon.

This important killer of Douglas-fir and associated species varies greatly in abundance, even over short distances. Most infection in present stands results from vegetative persistence of the fungus (*Poria weirii*) in dead roots of preceding stands. Most spread from tree to tree is by vegetative growth along the roots. Old-growth trees sometimes are infected, but damage consists principally of destruction of immature growing stock. Control is not yet possible, but economic impact can be reduced by the management practices recommended.

Childs, T. W.

1970. Laminated root rot of Douglas-fir in western Oregon and Washington. USDA Forest Serv. Res. Pap. PNW-102, 27 pp., illus. Pacific Northwest Forest & Range Experiment Station, Portland, Oregon.

This important killer of Douglas-fir and associated species varies greatly in abundance, even over short distances. Most infection in present stands results from vegetative persistence of the fungus (*Poria weirii*) in dead roots of preceding stands. Most spread from tree to tree is by vegetative growth along the roots. Old-growth trees sometimes are infected, but damage consists principally of destruction of immature growing stock. Control is not yet possible, but economic impact can be reduced by the management practices recommended.

Childs, T. W.

1970. Laminated root rot of Douglas-fir in western Oregon and Washington. USDA Forest Serv. Res. Pap. PNW-102, 27 pp., illus. Pacific Northwest Forest & Range Experiment Station, Portland, Oregon.

This important killer of Douglas-fir and associated species varies greatly in abundance, even over short distances. Most infection in present stands results from vegetative persistence of the fungus (*Poria weirii*) in dead roots of preceding stands. Most spread from tree to tree is by vegetative growth along the roots. Old-growth trees sometimes are infected, but damage consists principally of destruction of immature growing stock. Control is not yet possible, but economic impact can be reduced by the management practices recommended.

The mission of the PACIFIC NORTHWEST FOREST AND RANGE EXPERIMENT STATION is to provide the knowledge, technology, and alternatives for present and future protection, management, and use of forest, range, and related environments.

Within this overall mission, the Station conducts and stimulates research to facilitate and to accelerate progress toward the following goals:

1. Providing safe and efficient technology for inventory, protection, and use of resources.
2. Development and evaluation of alternative methods and levels of resource management.
3. Achievement of optimum sustained resource productivity consistent with maintaining a high quality forest environment.

The area of research encompasses Oregon, Washington, Alaska, and, in some cases, California, Hawaii, the Western States, and the Nation. Results of the research will be made available promptly. Project headquarters are at:

College, Alaska
Juneau, Alaska
Bend, Oregon
Corvallis, Oregon
La Grande, Oregon
Portland, Oregon
Roseburg, Oregon
Olympia, Washington
Seattle, Washington
Wenatchee, Washington

The FOREST SERVICE of the U.S. Department of Agriculture is dedicated to the principle of multiple use management of the Nation's forest resources for sustained yields of wood, water, forage, wildlife, and recreation. Through forestry research, cooperation with the States and private forest owners, and management of the National Forests and National Grasslands, it strives — as directed by Congress — to provide increasingly greater service to a growing Nation.